The Thoratec VAD has been used in a small number of children according to the manufacturer's database. As of May 2003, the Thoratec VAD had been used as a bridge to transplant in the United States in 8 children weighing less than 30 kg, but it had not been used in any children as a bridge to recovery. The smallest child was 17 kg.[5] Unlike extracorporeal membrane oxygenation, during the period of VAD support patients can be extubated and ambulate, allowing rehabilitation. There is no single solution for pediatric patients requiring mechanical support, but carefully selected pediatric patients may benefit from the Thoratec device as a bridge to transplant recovery. The current case demonstrates that, even in a relatively small child, the device can be removed without adverse effects on ventricular function despite apical cannulation.

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The bovine jugular vein conduit for right ventricular outflow tract reconstruction: A feasible alternative to homograft conduits?

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The bovine jugular vein conduit (BJVC) has been proposed for use in cardiac surgery for correction of congenital heart defects, either for right ventricular outflow tract (RVOT) reconstruction[1] or total cavopulmonary anastomosis completion in Fontan circulation.[2] Short-term and midterm results with the BJVC are controversial: satisfactory when used for RVOT reconstruction but rather disappointing for Fontan completion. In this study we report our early and late clinical results with BJVCs implanted in infants and children for RVOT reconstruction.

Clinical Summary

Between June 1999 and December 2002, 10 patients (7 male and 3 female patients) with a median age of 2 months (mean age, 13.7 ± 25.1 months) underwent surgical repair of congenital heart defects, including reconstruction of the RVOT with a BJVC (Contegra; Medtronic, Inc, Minneapolis, Minn).

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Patient demographics are detailed in Table 1.[3] Truncus arteriosus was the indication in 5 patients (with right ventricular hypoplasia in 1 patient), tetralogy of Fallot with pulmonary atresia in 2 patients, aortic arch interruption type B with severe aortic stenosis in 1 patient, double-outlet right ventricle with pulmonary atresia in 1 patient, and aortic stenosis in 1 patient. Four patients had undergone previous palliative procedures.

Results

There were 2 (20%) hospital deaths. One patient (patient 3) operated on for truncus arteriosus died suddenly on the 52nd postoperative day at home. The second patient (patient 5), who underwent a Ross-Konno procedure and a mitral valvuloplasty for aortic and mitral stenosis, died on the first postoperative day after an airway suction procedure. A third patient (patient 8), a baby girl operated on for truncus arteriosus type A-4 of Van Praagh classification, died of respiratory distress syndrome on the 20th postoperative day after extracorporeal membrane oxygenator assistance. Postmortem examination was performed in patients 3 and 5.

The remaining 7 patients were alive at the last follow-up. Echocardiographic data, recorded after a median time of 25 months (mean time, 24.3 ± 14 months), showed trivial incompetence of the BJVC in 3 patients and moderate incompetence in 1 patient. A transvalvular vein conduit peak gradient of greater than 30 mm Hg was observed in 2 patients. In 1 patient a peak gradient of greater than 30 mm Hg was detected at the level of the distal anastomosis between the conduit and the left pulmonary artery.

Morphologic investigation was performed in an unimplanted conduit that served as a control (Figure 1) and in the 2 conduits explanted at autopsy (Figure 2).

The unimplanted conduit showed a well-fixed tissue with leaflets consisting of a compact layer of collagen (fibrosa) and a layer

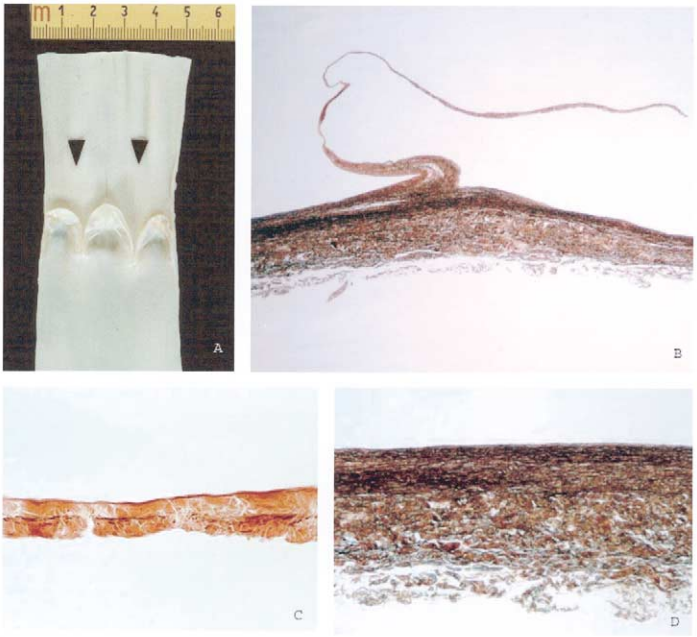

Figure 1. A, Control Contegra conduit: gross view of the bovine jugular vein with cusps in the native position. B, Histology of a leaflet and the vein wall (Weigert-Van Gieson staining). C, Close-up of the cusp, which mostly consists of collagen with loose elastic fibers (Weigert-Van Gieson staining). D, Close-up of the vein wall: note the parallel elastic lamellae orientation in the tunica media (Weigert-Van Gieson staining).

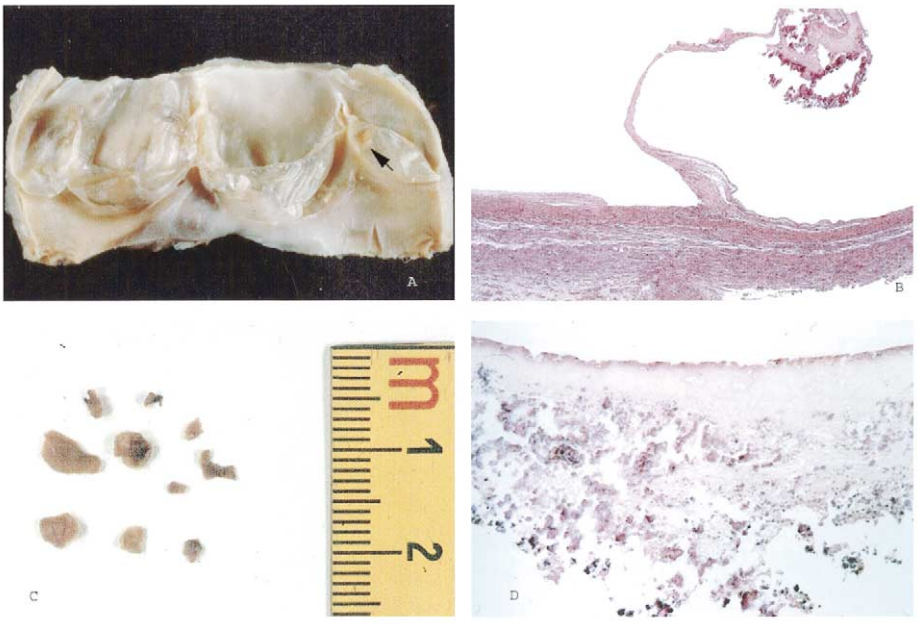

Figure 2. A, Contegra conduit in place for 52 days: gross view of the conduit at valve level explanted at autopsy. Note the fibrous sheeting and a nodular calcific deposit close to the commissure *(arrow)*. B, Histology of the nodular extrinsic calcification located at the free margin of the leaflet (hematoxylin and eosin staining). C, Calcium shells detached from the sinus side of the leaflets. D, Histology of the shells disclosing calcific deposits within the fibrous sheet (hematoxylin and eosin staining).

TABLE 1. Patient demographics

Patient no.	Sex/age at operation	Native pathology	Previous procedures
1	M/2 mo	Tetralogy of Fallot, absent pulmonary valve	No
2	F/12 mo	Aortic arch interruption type B, subaortic stenosis, VSD	Damus-Kaye Stensel procedure, modified B-T shunt
3	M/20 d	Truncus arteriosus, dysplastic truncal valve incompetence, patent ductus arteriosus	No
4	M/28 d	Truncus arteriosus, truncal valve incompetence, patent ductus arteriosus	No
5	M/5 mo	Aortic and mitral stenosis, left ventricular fibroelastosis	Percutaneous aortic valve plasty, surgical closure of right atrium-aorta iatrogenic communication
6	F/3 y	Truncus arteriosus, right ventricular hypoplasia	Cavopulmonary anastomosis, separation of the great arteries, modified B-T shunt
7	M/2 mo	Tetralogy of Fallot, pulmonary atresia, major aortic-pulmonary collateral arteries	RVOT reconstruction with a cryopreserved homograft, 8 mm; unifocalization of 2 collateral arteries at the right pulmonary ilum
8	F/10 d	Truncus arteriosus type A-4 of Van Praagh, truncal valve incompetence, aortic arch interruption, large PDA, ASD, VSD	No
9	M/10 d	Truncus arteriosus type 1–2, truncal valve incompetence, VSD, ASD	No
10	M/6 y	Double-outlet right ventricle, pulmonary atresia	B-T shunt, right pulmonary artery plasty

VSD, Ventricular septal defect; *RVOT,* right ventricular outflow tract; *B-T,* Blalock-Taussig; *PTFE,* polytetrafluoroethylene; *PDA,* patent ductus arteriousus; *ASD,* atrial septal defect.

of loose elastic fibers on the lumen side, with no evidence of spongiosa in between.

An endothelial lining was present on both sides. The vein wall showed a tunica media mostly consisting of parallel-oriented elastic lamellae, including smooth muscle cells and collagen fibers in between. The latter were quite prominent at the basal cuspal attachment. The adventitia exhibited scanty collagen and elastic fibers, loose extracellular matrix, and small vessels.

The explant in place at day 1 (patient 5) from a patient who died as a result of residual endocardial fibroelastosis, mitral stenosis, and pulmonary hypertension showed intact bovine vein cusps at gross examination and a thin fibrinous lining of both leaflet sides at histology caused by the endothelial detachment, with an otherwise intact vein wall.

The explant in place at days 52 (patient 3) from a patient who died suddenly at home with pulmonary edema most probably caused by a dysplastic incompetent truncal valve showed, to the naked eye, shells of calcium deposit on the leaflets within the sinuses, with a pinpoint calcification in the free margin close to a commissure. The shells, detached from the leaflets, were confirmed to be mineral deposits at radiography, which at histology appeared to occur within fibrous tissue. Von Kossa stain disclosed the calcific nature of mineralization extrinsic to the leaflet. Mild inflammatory infiltrates were observed in the subendothelium of the vein wall, as well as in the adventitia, with rare foreign body giant cells.

Discussion

The limited availability of homografts has forced surgeons to search for alternative tissue valved conduits for RVOT reconstruction. The BJVC (Contegra) is a glutaraldeyde-treated xenograft recently introduced on the market. Ichikawa and colleagues[4] reported excellent clinical results showing good tissue preservation, but on the other hand, Chang and associates[5] observed an inflammatory reaction involving either the leaflets or the conduit wall in BJVCs implanted in an animal model. Breymann and coworkers[6] reported a 27-month complete freedom from structural conduit deterioration, suggesting that the Contegra conduit might represent a promising alternative to homografts.

A report from Bové and colleagues[1] recently supported these findings. In their experience 41 patients with a mean age of 1.9 years who underwent RVOT reconstruction with a Contegra conduit showed satisfactory results, with excellent hemodynamic performances favorably comparable with those of pulmonary homografts. The major limit of the study was the shortness of follow-up. In their series they described one perioperative death but without further data on postmortem examination of the explanted conduit.

More recently, Schoof and associates[2] discouraged the BJVC use because of the catastrophic results obtained with such conduits when used for completion of the Fontan circulation. The authors'

Surgical procedure/date	Normal pulmonary valve orifice (mm)[3]	Outcome	Conduit pathology
VSD closure, RVOT reconstruction with Contegra, 12 mm; 6/10/1999	<8	Alive	
B-T shunt takedown, tunnel left ventricle-pulmonary artery with PTFE patch, RVOT reconstruction with Contegra 14, 9/12/1999	10.1	Alive	
VSD closure, RVOT reconstruction with Contegra, 12 mm; 2/29/2000	<8	Died (52 d postoperatively)	Extrinsic calcification on fibrous tissue overgrowth
VSD closure, RVOT reconstruction with Contegra, 12 mm; 3/21/2000	<8	Alive	
Ross-Konno operation; mitral valve plasty; RVOT reconstruction with Contegra, 12 mm; 5/16/2000	8.5	Died (1 d postoperatively)	Intact conduit, cusp fibrinous lining
One-and-a-half ventricle repair; VSD closure; RVOT reconstruction with Contegra, 16 mm; 9/19/2000	12.5	Alive	
VSD closure; left pulmonary artery plasty; RVOT reconstruction with a Contegra, 18 mm; 9/26/2000	12	Alive	
VSD closure; RVOT reconstruction with Contegra, 12 mm; 11/9/2000	<8	Died (20 d postoperatively)	Autopsy not performed
VSD closure; RVOT reconstruction with Contegra, 12 mm; 6/5/2002	<8	Alive	
B-T shunt take down; right and left pulmonary artery plasty; VSD closure; RVOT reconstruction with Contegra, 14 mm; 6/20/2002	14	Alive	

hypothesis was that glutaraldehyde cytotoxicity might be responsible for the high propensity for thrombosis by preventing the endothelial lining of the conduit. Moreover, they suggest that the cause of conduit thrombosis is the vein wall inflammatory reaction attributed to glutaraldehyde cytotoxicity, which is a more dangerous and proaggregating factor when in slow and nonpulsatile flow, as well as in Fontan circulation.

Our strategy was to use the Contegra conduit only in neonates and young infants (median age, 2 months).

In 9 of these patients, the implanted Contegra conduit was 4 mm larger than the expected normal pulmonary valve orifice, according to body surface area.[3] The 6-year-old patient, who received a 14-mm conduit, was affected by a severe pulmonary artery stenosis at the time of the operation, and therefore he also underwent a pulmonary artery plasty procedure. In this patient the plasty procedure was expected to be palliative, waiting for subsequent pulmonary artery diameter growth. In this way, at the time of that procedure, we decided not to use an oversized conduit because of the limitation of forward flow mostly as a result of pulmonary artery stenosis. To avoid larger right ventricular incision and therefore the unavoidable right ventricular failure, dilation, and geometric distortion, we usually prefer to use conduits that are not excessively oversized, reducing the right ventricular length incisions.

In conclusion, use of the BJVC was preferred to use of the homograft for RVOT reconstruction when its outgrowth was expected to anticipate structural degeneration. Unfortunately, the evidence of calcium deposits as early as 52 days after implantation in our postmortem specimens, which most probably occurred on thrombus deposits on fibrous sheets, prompted us to reconsider the pulmonary homograft as the conduit of choice.

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